Supplementary Figure Legends

Supplementary Figure S1. Northern blot analysis of *G0S2* expression in normal human tissues. A human multiple tissue northern blot was hybridized using a random-primed *G0S2* probe. The blot was normalized by loading equal amounts of total RNA in each lane.

Supplementary Figure S2. Quantitative RT-PCR analysis of gene expression in PFFs following TNFα treatment. PFFs were treated with TNFα (25 ng/ml) and at various time points monitored for expression of *G0S2*, *cIAP2, cIAP1* and *Bcl-xL* by qRT-PCR. *GAPDH* provided an internal normalization control, and the expression level obtained at time zero was assigned as 1. Error bars indicate SD.

Supplementary Figure S3. Time course of G0S2-mediated apoptosis and protein expression. *A*, H1299 cells were infected with Ad-G0S2 or, as a control, Ad-LacZ, and apoptosis was monitored at a series of time points following adenoviral infection. Error bars indicate SD. B, Immunoblot analysis monitoring expression of G0S2. Tubulin was monitored as a loading control.

Supplementary Figure S4. The G0S2(R57A, D58A) mutant protein retains mitochondrial localization. Immunofluorescence of Ad-G0S2- or Ad-G0S2(R57A, D58A)-infected H1299 cells stained with an α-HA antibody (left) or the mitochondrial marker Mitotracker (middle). Merged images are shown (right).

Supplementary Figure S5. Quantitative RT-PCR analysis monitoring *G0S2* knockdown in PFFs expressing a non-silencing (NS) shRNA or an shRNA directed against G0S2. Error bars indicate SD.

Supplementary Figure S6. *G0S2* is down-regulated in diverse human cancers. Expression of *G0S2* was analyzed in normal and cancer samples of brain (class 1, normal brain; class 2, oligodendroglioma); breast (class 1, normal breast; class 2, breast carcinoma); myeloma (class 1, normal bone marrow; class 2, smoldering multiple myeloma); ovary (class 1, normal ovary; class 2, ovarian endometrioid adenocarcinoma) and renal (class 1, normal kidney tissue; class 2, clear cell renal cell carcinoma). Data were obtained through the Oncomine database; a Student's T-test, performed directly though the Oncomine 3.0 software, showed the difference in gene expression between the samples was significant.

Supplementary Figure S7. ShRNA-mediated knockdown of G0S2 promotes oncogene-induced transformation. (Left) Colony formation assay. Mouse embryonic fibroblasts stably expressing a non-silencing (NS) or G0S2 shRNA were infected with a retrovirus encoding the oncogenes E1A and ras, and 3 weeks later cells were fixed and stained with methylene blue to visualize transformed foci. (Right) RT-PCR analysis monitoring target gene knockdown in MEFs expressing a NS or G0S2 shRNA. *Actin* was monitored as a loading control. *G0S2* expression was quantified; the level of *G0S2* mRNA in G0S2 knockdown cells is given relative to that observed in control cells, which was assigned as 1.